

reaction is in progress, but it is not clear against what antigen it is directed. It is just possible that the disease could result from some form of chronic immunological reaction (a situation analogous to the NZB mice with autoimmune haemolytic anaemia and to experimental animals with chronic graft-versus-host disease, both of which have an increased incidence of tumours of the reticuloendothelial system (East and Prosser, 1967; Schwartz, 1968), but there is little evidence to support such a view.

The changes in Hodgkin's disease could represent a reaction against an undiscovered associated infection. Such an explanation is impossible to refute, but most of the patients studied had no fever and no clinical or laboratory changes indicating infection. Nevertheless, a continuing search for viral infections in this disease may well prove rewarding, particularly in view of the recent work of Epstein *et al.* (1964), Klein *et al.* (1968), and Henle *et al.* (1966, 1968) showing a herpesvirus infection to be an invariable accompaniment of Burkitt's lymphoma and infectious mononucleosis. Patients with Hodgkin's disease are known to be particularly prone to certain viral, bacterial, fungal, and parasitic infections (Aisenberg, 1966).

Immunological reactions have now been found in a number of human malignant diseases, but it is not known whether the presence of such a reaction is merely a consequence of tumour degeneration rather than a factor in host resistance. The most interesting explanation is that the reactive lymphoid cells are present in response to the putative tumour-specific antigen, and hence the disease, but the possibility remains that they represent a reaction to tumour products.

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## REFERENCES

- Aisenberg, A. C. (1966). *Cancer Research*, **26**, 1152.  
 Bouroncle, B. A. (1966). *Blood*, **27**, 544.  
 Brown, R. S., Haynes, H. A., Foley, H. T., Godwin, H. A., Berard, C. W., and Carbone, P. P. (1967). *Annals of Internal Medicine*, **67**, 291.  
 Coulson, A. S., and Chalmers, D. G. (1967). *Immunology*, **12**, 417.  
 Crowther, D., Fairley, G. H., and Sewell, R. L. (1967). *Nature*, **215**, 1086.  
 Crowther, D., Fairley, G. H., and Sewell, R. L. (1969). *Journal of Experimental Medicine*, **129**, 849.  
 East, J., and Prosser, P. R. *Proceedings of the Royal Society of Medicine*, **60**, 823.  
 Epstein, M. A., Achong, B. G., and Barr, Y. M. (1964). *Lancet*, **1**, 702.  
 Henle, W., Hummeler, K., and Henle, G. (1966). *Journal of Bacteriology*, **92**, 269.  
 Henle, G., Henle, W., and Diehl, V. (1968). *Proceedings of the National Academy of Sciences*, **59**, 94.  
 Klein, G., Pearson, G., Henle, G., Henle, W., Diehl, V., and Niederman, J. C. (1968). *Journal of Experimental Medicine*, **128**, 1021.  
 Klima, R. (1952). *Wiener Zeitschrift für innere Medizin und ihre Grenzgebiete*, **33**, 125.  
 Kuper, S. W. A., and Bignall, J. R. (1964). *Lancet*, **1**, 1412.  
 Peters, M. V., Alison, R. E., and Bush, R. S. (1966). *Cancer*, **19**, 308.  
 Schwartz, R. S. (1968). Plenary Session Papers of the 12th Congress of the International Society of Hematology, New York, 1968.

## Prevention of Prolonged Labour

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**S**ummary: A prospective study of 1,000 consecutive primigravid deliveries has shown that active management in labour can ensure that every woman is delivered within 24 hours. Emphasis is laid on the importance of a correct initial diagnosis of labour based on objective criteria. Amniotomy followed by oxytocin infusion is advocated to simulate the progress of normal labour unless this is evident from an early stage.

Oxytocin, the dose of which is limited only by foetal distress, cannot be used effectively unless three popular fallacies are rejected. Firstly, that prolonged labour is often an expression of cephalo-pelvic disproportion; secondly, that oxytocin may rupture the primigravid uterus; and, thirdly, that there is a valid therapeutic distinction between hypotonic and hypertonic uterine action.

Stimulation, properly supervised, is safe to mother and child, it eliminates the problem of occipitoposterior position, results in a sharp decline in forceps delivery, and obviates the need for massive analgesia.

### Introduction

Prolonged labour presents a picture of mental anguish and physical morbidity which often leads to surgical intervention and may produce a permanent revulsion to childbirth, expressed by the mother as voluntary infertility; it constitutes a danger to the survival and subsequent neurological development of the infant (Jeffcoate *et al.*, 1952; *Lancet*, 1963). The harrowing experience is shared by relatives, and by doctors and nurses to the extent that few complications so tarnish the image of obstetrics.

There have been many publications on the subject which have been concerned mainly with aetiology, complications, and treatment (Hawkins and Nixon, 1957; Garrett, 1960; Barr, 1964). When prevention has been considered the value of stimulation has been presented as controversial or the application has been restricted by excessive caution (Garland, 1961; Schulman, 1966). Because no course of management which is not qualified by serious limitations has been described, it is not surprising that prolonged labour continues to be a problem. The purpose of this paper is to show that prolonged labour can be prevented by effective stimulation. In the process it is emphasized that some of the conventions of delivery ward practice must be abandoned.

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During recent years an active approach to labour has been adopted progressively at the National Maternity Hospital, and as the definition of prolonged labour was reduced from 48 to 36 and then to 30 hours the incidence continued to fall. The standard was finally reduced to 24 hours, and the claim that prolonged labour can be prevented is based on a prospective study of 1,000 consecutive primigravid deliveries, all of which were under our personal supervision.

### Material and Methods

The analysis which follows is of 1,000 consecutive primigravidae delivered between 1 January and 16 September 1968. Multigravidae were excluded to avoid diluting the results with cases in which there is a very low incidence of prolonged labour. There were 2,423 multigravidae delivered during the same period and none was in labour for 24 hours.

The duration of labour was estimated from the time labour began until the baby was born, and, like all such estimates, was subject to personal interpretation because there is no precise definition of when labour begins. The emphasis was on objective evidence, and painful uterine contractions were not accepted as conclusive unless there was a show, spontaneous rupture of the membranes, or dilatation of the cervix. A firm decision was reached in each case within a few hours of admission, and on this decision depended the subsequent management. Labour was classified as prolonged when it exceeded 24 hours.

When a decision was made that labour had started, an active policy was pursued. Unless dilatation of the cervix was progressive the membranes were ruptured, and if this did not accelerate progress an intravenous oxytocin infusion was begun and continued until the placenta was delivered. Oxytocin was never infused when the membranes were intact. The concentration of oxytocin was constant at 10 units per litre of 5% dextrose solution. The rate of infusion was regulated to ensure dilatation of the cervix and was limited only by signs of foetal distress. Pethidine was the only analgesic used, and it was not given until progress was established. Prenatal relaxation classes were suspended during the survey and did not contribute to the results.

The possible adverse effects of active management on the foetus were measured by examining the perinatal deaths and the infants who showed clinical evidence of residual brain damage at discharge from hospital; the effects on the mother were measured by examining the caesarean sections and operative vaginal deliveries.

### Results

In this series of 1,000 consecutive primigravid deliveries there was one woman in labour for more than 24 hours. This patient was admitted with ruptured membranes and painful uterine contractions. Oxytocin was started 11 hours later because the cervix had not dilated, and the baby was delivered by forceps 28 hours after admission.

In the series there were also eight emergency admissions with a diagnosis of prolonged labour. Three patients were not in labour though they had been given relatively large doses of analgesics; after treatment for dehydration and ketosis labour was induced and they were delivered within 24 hours of admission. The other five patients were stimulated with oxytocin and delivered within nine hours of admission.

Artificial rupture of the membranes was performed to accelerate labour in 119 cases. Progress was satisfactory in 84 cases with the following results: 68 were delivered within 6 hours, 13 within 12 hours, and 3 within 18 hours. Progress was unsatisfactory in the other 35 cases, which were then stimulated with oxytocin with the following results: 29 were delivered within 6 hours, 3 within 12 hours, 2 within 18 hours,

and 1 within 24 hours. In 85 additional cases oxytocin was administered to accelerate labour after spontaneous rupture of the membranes or after surgical induction of labour, with the following results: 68 were delivered within 6 hours, 14 within 12 hours, and 3 within 18 hours. The number of women, therefore, who were treated to accelerate the progress of labour was 204 (20.4%). These may be summarized thus: 84 had artificial rupture of membranes, 35 had artificial rupture of membranes and oxytocin, and 85 had oxytocin only.

### Perinatal Deaths

There were 25 perinatal deaths in 1,010 infants, which included 10 sets of twins (2.5%). The circumstances were as follows: 8 had congenital malformation, 10 died before labour, 3 died during labour, and 4 died after birth. Necropsy was performed in every case. The malformations were two cases of congenital heart disease, two of anencephalus, two of hydrocephalus, and one each of renal agenesis and oesophageal atresia. In the cases in which death occurred before labour the foetus was born macerated on all but two occasions, when death was due to abruption of the placenta. Two neonatal deaths followed elective caesarean section. Deaths from congenital anomaly, deaths before labour, and neonatal deaths after elective caesarean section are not influenced by labour and therefore are not considered further. There remain five infants who died after labour began.

The foetus died during labour on three occasions and in each case death was due to hypoxia before caesarean section could be undertaken for foetal distress. None of these cases had been treated with oxytocin. Amniotomy had been performed to accelerate labour in one instance. The liquor was stained with meconium; the foetal heart was slow after one hour and ceased 15 minutes later. Necropsy showed extensive subserous petechial haemorrhages and inhalation of meconium.

There were two neonatal deaths. The only neonatal death which occurred after treatment to accelerate labour was in a second twin. The cervix was fully dilated when the mother was admitted with complete secondary inertia. Oxytocin was given because the first twin presented as a breech. Spontaneous delivery occurred one hour later. The second twin was delivered by forceps for profound distress and died within minutes. Necropsy showed subserous petechial haemorrhages and inhalation of meconium.

### Brain Damage

Four infants showed clinical evidence of residual damage to the central nervous system. Measures to accelerate labour had been taken in one case. The maturity was 42 weeks, no liquor drained at amniotomy after four hours in labour, and an oxytocin infusion was added after an interval of six hours. No abnormality of the foetal heart was detected, but meconium was present at delivery one hour later. An asphyxiated baby responded to endotracheal insufflation after five minutes but subsequently suffered cerebral irritation and motor seizures.

### Caesarean Section

There were 40 caesarean sections (4%). The operation was performed before labour in 28 cases and during labour in 12. The indications for caesarean section during labour were: disproportion in 5, foetal distress in 3, brow presentation in 1, breech presentation in 1, abruptio placentae in 1, and prolapsed cord in 1. Two patients had been treated to accelerate labour. In one case the membranes were ruptured to induce labour for slight antepartum haemorrhage. Meconium drained as labour began 18 hours later; an oxytocin infusion was started to stimulate progress, but caesarean section was performed for

toetal distress. The indication for caesarean section in the other case was prolapse of the cord at amniotomy.

### Operative Vaginal Delivery

There were 189 forceps or ventouse deliveries (18.9%). The incidence in primigravidae during the two previous years was 30.3 and 23.8%. As the incidence of forceps or ventouse is usually calculated to include multigravidae as well as primigravidae, the comparable figures for this hospital in the three years 1966–8 were 12, 9.5, and 6.7% of all patients.

### Discussion

The progress of labour should be regulated to ensure that every woman is delivered within 24 hours. The results obtained in the present series of 1,000 consecutive primigravidae show that this can be achieved provided the obstetrician assumes direct responsibility and forsakes the role of passive observer for that of active director, controlling the course of labour instead of waiting in the hope that it may conclude within a reasonable period of time. The method described is completely opposed to that of Friedman and Sachtleben (1961), who urged an approach even more conservative than is currently practised.

One woman was in labour for more than 24 hours after admission to hospital and she alone exhibited the clinical syndrome of prolonged labour. She was dehydrated and exhausted, retained urine, and vomited, on the last occasion during forceps delivery. This failure stemmed from indecision, which is the most important causative factor in prolonged labour. An unequivocal diagnosis of labour was not made for several hours after admission, stimulation was then unduly delayed, and when applied it was not sufficient to produce an effective response.

In adopting an active role the first duty of the obstetrician is to decide whether or not a woman is in labour. This decision must not be surrendered to the mother, assuming that she is in labour simply because she says so. It must be recognized that a primigravida, particularly with scant knowledge and no experience, is not qualified to make the crucial decision on which subsequent management depends. This is where the fundamental mistake is often made. Uterine inertia is diagnosed before labour has started, and this results in unnecessary alarm and treatment (Jeffcoate, 1961). The present series included eight emergency admissions for prolonged labour, and three of these women, though suffering from dehydration, ketosis, and exhaustion, were not in labour.

A diagnosis can be made in most cases within a few hours of admission. When it is decided that a woman is not in labour analgesic drugs should not be given and she should be removed to the distractions of an antenatal ward. The paramount importance of objective evidence in the diagnosis of labour should be explained to the patient. This approach proved very successful in the present series, in which some women were allowed home after a lapse of 24 hours; the others, meanwhile, were returned to the delivery unit in labour. A decision that a patient was not in labour did not always prove to be correct, but what was important was that a decision on management had been made.

When a decision is made that a woman is in labour progress must be kept under constant review. This can only be achieved by making a formal round of the delivery unit at regular intervals every day, and especially late at night. Decisions in doubtful cases may be deferred until the next round. The membranes should be ruptured at an early stage if dilatation of the cervix is not progressive. This was done on 119 occasions in the present series, with a satisfactory response in 70% of cases, which required no further treatment. No harm resulted to any mother or child, and there was no evidence that

rupture of the membranes retarded labour, as suggested by Friedman and Sachtleben (1962).

An intravenous infusion of oxytocin should be started if progress is not satisfactory after rupture of the membranes. This was done in 120 cases in the present series, 80% of which delivered within six hours and 95% within 12 hours. A standard concentration of oxytocin, 10 units per litre, was used in all cases and only the rate of infusion was changed. A standard solution eliminates one of two variable factors and therefore increases the margin of safety. Progress should be measured by dilatation of the cervix, not by the subjective reaction of the patient. Too often women are given to understand that not only are they expected to diagnose the onset but also to interpret the progress of labour. To subordinate the rate of infusion to the reaction of the patient is to allow her to control the drip.

The initial rate of infusion was 5 drops, and subsequently there was no theoretical restriction, though it never proved necessary to exceed 80 drops (50 mU.) per minute. In practice the only limiting factor was foetal distress. Goldman (1959), who expressed similar views on oxytocin, achieved delivery within 12 hours in 85% of cases compared with 95% in the present series. This discrepancy may have been caused by his use of a more dilute concentration, 4 units per litre, at a rate which rarely exceeded 30 drops per minute. The only perinatal death which occurred after oxytocin was in a second twin. We agree with Goldman that oxytocin infusion should not endanger the life of the child. The contrary should be true, because with proper supervision it converts abnormal into normal labour.

No review of clinical management in obstetrics would be complete without reference to residual brain damage, which is the ultimate failure for the child. There were four suspected cases in the present series of firstborn infants (0.4%), and all were the result of hypoxia. Oxytocin had been infused to accelerate labour in one case which was complicated by post-maturity. No conclusion is offered, but it is suggested that the evaluation of a method of treatment must take into account considerations beyond the preservation of life.

Oxytocin cannot be used effectively unless there is a confident rejection of three popular fallacies. These are that prolonged labour is often an expression of cephalo-pelvic disproportion (Friedman and Sachtleben, 1962), that oxytocin may rupture the primigravid uterus (Garland, 1961), and that there is a valid therapeutic distinction between hypotonic and hypertonic uterine activity (Jeffcoate, 1961; Johnson, 1961). To these may be added a mistaken concern with occipitoposterior position.

In this hospital the number of cases in which a diagnosis of disproportion is made has fallen steadily since the active approach to labour was adopted. There were five cases in the present series of 1,000 primigravid deliveries (0.5%). The reason for this reduction is that trial of labour is not permitted to fail from inefficient uterine action.

The primigravid uterus is almost immune to rupture except by manipulation. Goldman (1959) reviewed the literature for reports of rupture of the uterus after oxytocin infusion and found no case in a primigravida. In 100 consecutive cases of rupture of the uterus treated in the Dublin Maternity Hospitals, which did not include caesarean section scars, there were four primigravidae; two were caused by manipulation, one by hydrocephalus, and there was no explanation in one case (O'Driscoll, 1966). In a clinical study of 181 consecutive cases of ruptured uterus reported by Trivedi *et al.* (1968) no patient was a primigravida.

No distinction was made in the present series between hypotonic and hypertonic uterine activity, and oxytocin proved equally effective in both circumstances. This is completely at variance with the experience of Jeffcoate (1961), who found that oxytocin often makes matters worse when given in labour

complicated by incoordinate activity. It is our experience that the correct treatment of incoordinate action is to increase the rate of oxytocin infusion and not the dose of analgesic drugs.

The position of the foetal head did not influence the outcome of labour in any case in this series, and caesarean section was not performed for occipitoposterior or transverse positions. This contrasts with the experience at the Simpson Memorial Maternity Pavilion (1967), where 71 caesarean sections were performed during a period of one year in cases of occipitoposterior or transverse position and in one-half the indication was disordered uterine action. In our experience position of the foetal head is not a matter of consequence when effective uterine contractions are maintained.

The introduction of a policy of controlled labour was followed by a sharp reduction in the incidence of forceps delivery in primigravidae from 30.3 to 18.9% over a period of two years. The greatest proportionate reduction was under the heading foetal distress, which was the indication for 42.5% of forceps deliveries in 1966 and for 34% in 1968. Difficult cases of rotation and high application are now rarely seen. There are two reasons for this: mothers are not exhausted when they enter the second stage of labour and effective contractions are maintained until delivery.

A generous use of analgesic drugs is characteristic of the passive approach to labour, but comparatively little attention is paid to the effect of these drugs on behaviour. The situation is created in which women demand more and more drugs simply because they are progressively confused. It has been suggested that those who seek oblivion should be given it and that a patient should be encouraged to complain of pain during childbirth until it is relieved to her satisfaction, to achieve which even heroin is recommended (*British Medical Journal*, 1966; Beazley *et al.*, 1967). We are satisfied that women in labour regard heavy sedation as a poor substitute for the promise of early delivery. Furthermore, it is our experience that when labour is slow women often complain of intolerable discomfort, but when progress is stimulated the nature of the discomfort changes and oxytocin is more effective than analgesia in the relief of pain. Pethidine was the only drug used in the present series, and, though it was subject to critical appraisal, there was no policy of restriction. A measure of the reduced need for analgesia when an active policy is pursued is that only eight patients (0.8%) received 200 mg. of pethidine.

Prolonged labour is so much less frequent in multigravidae that the incidence should be expressed in terms of primigravid deliveries only. This should apply also to subjects which are closely related to inefficient uterine action, such as cephalopelvic disproportion, occipitoposterior position, and forceps delivery. This practice was not followed by Turnbull and Anderson (1968) in their reference to acceleration of labour with oxytocin, which makes their otherwise detailed observations difficult to interpret. In the case of a multigravida when progress is slow it is far more likely to be a product of

obstructed labour, due to a foetal cause, which must be excluded before stimulation is applied. During the period of the present study, 2,423 multigravidae were delivered, none was in labour for 24 hours, and the incidence of forceps delivery was 1.8%.

Active management in labour can be conducted only on the basis of individual attention which requires a delivery unit, in effect a special care unit, in which no patient is left unattended and skilled nursing attention and medical supervision are always at hand. These requirements cannot be met in domiciliary practice. When progressive dilatation of the cervix is not occurring early transfer to hospital is advisable.

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ADDENDUM.—The four infants who showed evidence of brain damage have been reviewed since this paper was submitted for publication. At 6 months of age three infants had cerebral palsy and one was normal. The normal infant was the case in which oxytocin had been given.

## REFERENCES

- Barr, W. (1964). *British Medical Journal*, **1**, 1427.  
 Beazley, J. M., Leaver, E. P., Morewood, J. H. M., and Bircumshaw, J. (1967). *Lancet*, **1**, 1033.  
*British Medical Journal*, 1966, **2**, 1282.  
 Friedman, E. A., and Sachtleben, M. R. (1961). *Obstetrics and Gynecology*, **17**, 566.  
 Friedman, E. A., and Sachtleben, M. R. (1962). *Obstetrics and Gynaecology*, **19**, 576.  
 Garland, G. W. (1961). *Practitioner*, **186**, 578.  
 Garrett, W. J. (1960). *Medical Journal of Australia*, **2**, 481.  
 Goldman, L. (1959). *Journal of Obstetrics and Gynaecology of the British Empire*, **66**, 382.  
 Hawkins, D. F., and Nixon, W. C. W. (1957). *Journal of Obstetrics and Gynaecology of the British Empire*, **64**, 641.  
 Jeffcoate, T. N. A. (1961). *Lancet*, **2**, 61.  
 Jeffcoate, T. N. A., Baker, K., and Martin, R. H. (1952). *Surgery, Gynecology, and Obstetrics*, **95**, 257.  
 Johnson, R. E. (1961). *Obstetrics and Gynecology*, **18**, 468.  
*Lancet*, 1963, **2**, 391.  
 O'Driscoll, K. (1966). *Proceedings of the Royal Society of Medicine*, **59**, 65.  
 Schulman, H. (1966). *American Journal of Obstetrics and Gynecology*, **95**, 732.  
 Simpson Memorial Maternity Pavilion, Edinburgh (1967). *Medical and Clinical Report*, p. 52.  
 Trivedi, R. R., Patel, K. C., and Swami, N. B. (1968). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, **75**, 51.  
 Turnbull, A. C., and Anderson, A. B. M. (1968). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, **75**, 32.